Catatonia: Where to Look When ECT Fails

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BACKGROUND

It is well accepted that catatonia has a high prevalence in autoimmune encephalitis and there is support in the literature for incorporating ECT as standard treatment for these patients when immunomodulation therapies are insufficient (Tanguturi, 2019). We present the case of a patient with a history of bipolar I disorder who presented with catatonia refractory to ECT and aggressive medical management. Due to her prior psychiatric history, an autoimmune etiology was not seriously considered at the OSH prior to embarking on ECT. This case report highlights the importance of considering autoimmune catatonia regardless of psychiatric history; and proposes further research into a potential link between anti-basal ganglia antibodies and autoimmune catatonia.

IVIg

Intravenous immune globulin (IVIg), a derivative of donated plasma, is used in the treatment of various autoimmune disorders. It is presently the first-line immunotherapy for autoimmune encephalitis despite the lack of literature supporting its efficacy (Lee et al., 2022). The exact mechanism of action of IVIg is not fully understood, however, various mechanisms have been proposed including immunomodulation, inhibition of autoantibodies, reduction of pro-inflammatory cytokines and complement system suppression. The specific mechanism involved in autoimmune encephalitis may involve the inhibition of naive CD4 T cell differentiation into encephalitogenic cells (Othy S et al., 2013)

Case Description

A 65-year-old female with bipolar disorder who presented to Baptist Health with altered mental status, rigidity, and decreased responsiveness was admitted for severe catatonia.The cause of her catania had been attributed to her bipolar disorder after unremarkable LP and MRI. After 32 days of failed medical management, she was transferred to UAMS for ECT with subsequent treatment noted below.

TIMELINE

Day 1 - 32: Presented to Baptist Health with catatonia BFCRS score 37. Treatment with IV Ativan 2.5 mg QID and memantine 5 mg BID. Dose adjustments made, Ativan doses as high as 25 mg daily
Day 33: Transferred to UAMS, started Ativan 3 mg IV q6h and Memantine 5 mg BID
Day 38: Started ECT and Lithium 300 mg daily
Day 45: Ativan increased to 3.5 mg q4h IV
Day 46: Lithium increased to 600 mg daily
Day 50: Minor improvements in rigidity and mentation were noted
Day 51: Ativan decreased to 2 mg IV q4h
Day 75: ECT d/c'd after 15 total sessions with minimal improvement
Day 81: Autoimmune encephalitis workup ordered; notable for GAD65 ab borderline elevation, cunningham panel notable for anti-D1 receptor ab and anti-tubulin ab. CSF studies notable for 5 WBCs
Day 82: Lithium discontinued, VEEG showed generalized background slowing
Day 90-94: Treated with IVIg 0.4g/kg/day for 5 days with significant improvement in speech, rigidity, motor control
Day 100-110: 5 sessions of plasmapheresis
Day 128: Patient discharged with BFCRS score 3

DISCUSSION

Our case confirms that successful treatment of catatonia must take into account the underlying etiology of the catatonia. For our patient, standard treatment of catatonia was ineffective until the autoimmune process was appropriately addressed. Interestingly, she never had a positive antibody on the Mayo ENC 2 panel identified, yet her catatonia responded robustly to immunomodulation. Diagnostically, probable seronegative autoimmune encephalitis would be the most unifying diagnosis. In the literature, cortico-striato-thalamo circuits are implicated in the pathophysiology underlying catatonia (Northoff, 2002), which is why we opted to look for presence of anti-basal ganglia antibodies. The Cunningham panel is well known in the PANDAS literature but further research is needed to explore its utility in patients with catatonia caused by seronegative autoimmune encephalitis.

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